5. Levréro F, Gatti S, Gautier-Hion A, Ménard N. Yaws disease in a wild gorilla population and its impact on the reproductive status of males. Am J Phys Anthropol. 2007;132:568–75. http://dx.doi.org/10.1002/ajpa.20560
6. Knauf S, Batamuzi EK, Mlengeya T, Kilewo M, Lejora IA, Nordhoff M, et al. *Treponema* infection associated with genital ulceration in wild baboons. Vet Pathol. 2012;49:292–303. http://dx.doi.org/10.1177/0300985811402839
7. Harper KN, Fyumagwa R, Hoare R, Wambura P, Coppenhaver D, Sapolsky R, et al. *Treponema pallidum* infection in the wild baboons of East Africa: distribution and genetic characterization of the strains responsible. PLoS ONE. 2012;7:e50882. http://dx.doi.org/10.1371/journal.pone.0050882
8. Zobaníková M, Strouhal M, Mikalová L, Cejková D, Ambrožová L, Pospíšilová P, et al. Whole genome sequence of the *Treponema* Fribourg-Blanc: unspecified simian isolate is highly similar to the yaws subspecies. PLoS Negl Trop Dis. 2013;7:e2172. http://dx.doi.org/10.1371/journal.pntd.0002172
9. Smith JL, David NJ, Indgin S, Israel CW, Levine BM, Justice J Jr, et al. Neuro-ophthalmological study of late yaws and pinta. II. The Caracas project. Br J Vener Dis. 1971;47:226–51.
10. Thomson JG, Lamborn WA. Mechanical transmission of trypanosomiasis, leishmaniasis, and yaws through the agency of non-biting haematophagous flies. BMJ. 1934;2:506–9. http://dx.doi.org/10.1136/bmj.2.3845.506

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Porcine Hokovirus in Domestic Pigs, Cameroon

To the Editor: Since 2005, new parvoviruses forming a novel genus of the proposed name *Partetravirus*, within the subfamily *Parvovirinae*, have been described (*1*). Human parvovirus 4 (PARV4) with 3 different genotypes globally infects humans (*2*). A related porcine virus, hokovirus (HoV or porcine partetravirus), was found in wild boar and domestic pig populations in Germany, Romania, China, and the United States, with prevalences of 12%–47%, forming 1 common genotype (*3*–*6*). Prevalence figures from sub-Saharan Africa are not available. Furthermore, no information about possibly region-associated genotypes is available for porcine HoV, although it is for human PARV4 from the same genus. We therefore used samples (collected during February–March 2012) from a study investigating hepatitis E virus (HEV) in pigs from Cameroon (*7*) to analyze the occurrence of porcine HoV in pigs in Africa and to determine the respective genotype.

Viral DNA was extracted from liver samples by using the RTP DNA/RNA Virus Mini Kit II (STRATEC-Molecular, Berlin, Germany) according to the manufacturer's instructions. DNA samples were pooled, with each pool containing 3 different samples. A total of 94 pooled samples from 282 animals originating from 3 districts in Cameroon (Doula, Yaoundé, and Bamenda) were investigated by using quantitative real-time PCR (*3*,*7*). Samples from pools that tested positive were analyzed individually.

We detected HoV in 65 (69%) of the 94 pooled samples: 2 (15%) of 13 from Bamenda, 39 (70%) of 56 from Douala, and 24 (96%) of 25 from Yaoundé. We used an online tool to estimate the individual prevalence from pooled samples for fixed pool size and perfect test with exact 5% upper and lower CIs (http://epitools.ausvet.com.au/content.php?page = PooledPrevalence). A pool size of 3 with a total of 94 pooled samples and 65 positive samples resulted in an estimated general prevalence of 32.4% (95% CI 27%–39%). For Bamenda, the estimated prevalence was 5.4% (95% CI 1%–16%); for Douala, 32.8% (95% CI 25%–41%); and for Yaoundé, 65.8% (95% CI 44%–87%).

From 94 positive pools, a total of 184 samples were available for individual testing: 6 from Bamenda, 110 from Douala, and 68 from Yaoundé; 12 were missing. Using the results from the negative tested pools and the individual testing, we found an estimated general prevalence of 47% (128/270). The regional prevalence was 10% (4/39) for Bamenda, 41% (65/160) for Douala, and 83% (59/71) for Yaoundé.

These prevalences are higher than the estimates, but lie within the regional estimates within the range of the CI determined with the online tool. The discrepancy in the total prevalence might be due to the missing samples for the individual testing. Our results show that pooled sample testing can yield a good approximation of the actual prevalence, at least for settings in Africa. The varying prevalence and inhomogeneous regional distribution of porcine HoV correspond to previous findings from Europe, China, and the United States in wild boar and domestic pigs (*3*,*5*,*6*). Overall, no general defined pig-breeding program is in place in Cameroon. Douala and Yaoundé are the main markets for pig trade. Yaoundé, the main town for pig purchase and slaughter, gets live pigs from northwestern (Bamenda), western, and northern Cameroon, and Douala receives pigs from northwestern (Bamenda), western, and southwestern Cameroon. To fully understand the observed regional prevalences, the presence of HoV needs to be investigated in detail in the southwest, west, and north, where intensive farming systems are in place and pig farming is of economic importance.

Near full-length genome data were generated from 3 positive samples, and partial sequence information was retrieved for 8 additional samples (Figure) as described (*3*). The phylogenetic analysis showed a very close relation, with 98%–99% homology between the porcine HoV isolates from Cameroon, Europe, the

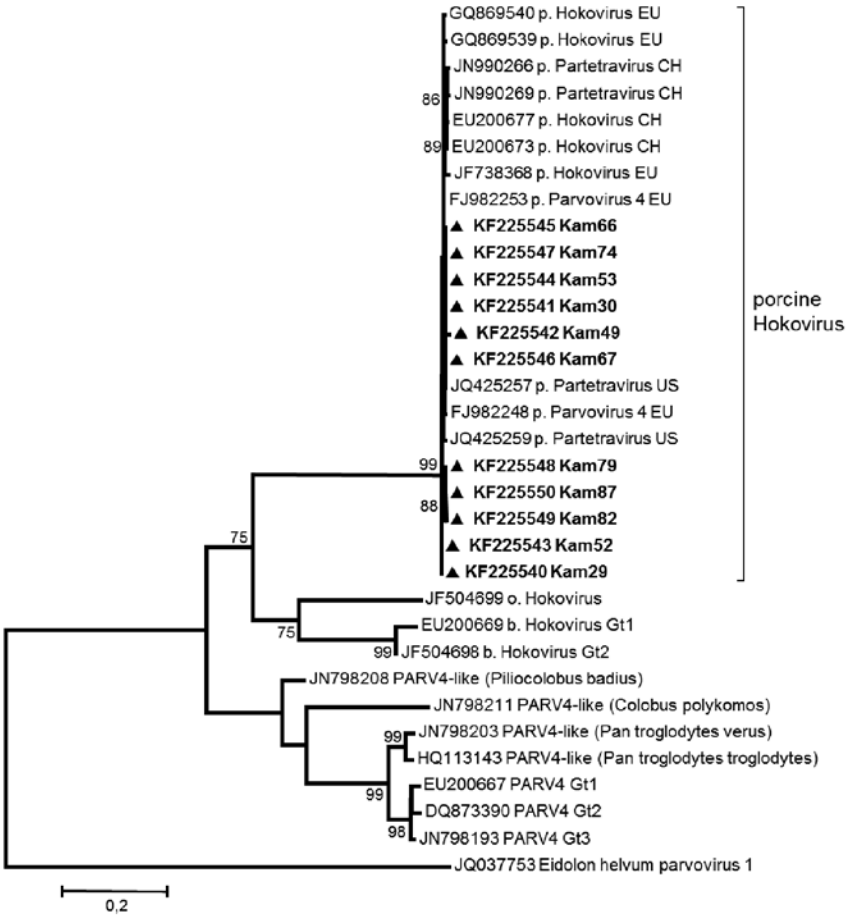

Figure. Phylogenetic tree of near full-length (4.7 kbp) and partial sequences (open reading frame 2, 0.4–1.9 kbp) of porcine hokovirus (HoV)/partetraviruses (PtV) created by using MEGA5.1 (www.megasoftware.net) with the maximum-likelihood method (GTR+G+I) and bootstrap analysis of 500 resamplings. New sequences from Cameroon are shown in **boldface**. EU, Europe; CH, China; US, United States; Gt, genotype; PARV4, parvovirus 4. Scale bar indicates nucleotide substitutions per site.

United States, and China. In contrast to bovine HoV or PARV4 in humans and chimpanzees, only 1 porcine genotype has been observed worldwide, which might point to 1 common ancestor (*8*,*9*). Because the liver samples were taken from European landrace or cross-bred pigs, the common ancestor of HoV might have originated from Europe. Pigs chronically infected with HoV could have been imported from Europe to Cameroon during the extensive global industrial farming and pig trade in the last century. The detection of the European HEV genotype (gt) 3 instead of the "traditional" African gt1 and gt2 in these pigs supports this hypothesis (*7*,*10*). Nevertheless, the high grade of homology of porcine HoV found worldwide suggests a highly species-specific virus with a low mutation frequency. To study the circulating genotypes in Africa and to generate hypotheses on the influence of trade, researchers need to college data from other African countries. However, data on HoV in wild animals, such as warthogs and pot-bellied pigs, are limited and need to be addressed to elucidate the genomic intravariability and intervariability of this new family of parvoviruses.

Generated sequences were submitted to GenBank under accession nos. KF225540–KF225550.

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References

1. Jones MS, Kapoor A, Lukashov VV, Simmonds P, Hecht F, Delwart E. New DNA viruses identified in patients with acute viral infection syndrome. J Virol. 2005;79:8230–6. http://dx.doi.org/10.1128/JVI.79.13.8230-8236.2005
2. Simmonds P, Douglas J, Bestetti G, Longhi E, Antinori S, Parravicini C, et al. A third genotype of the human parvovirus PARV4 in sub-Saharan Africa. J Gen Virol. 2008;89:2299–302. http://dx.doi.org/10.1099/vir.0.2008/001180-0
3. Adlhoch C, Kaiser M, Ellerbrok H, Pauli G. High prevalence of porcine Hokovirus in German wild boar populations. Virol J. 2010;7:171. http://dx.doi.org/10.1186/1743-422X-7-171
4. Cadar D, Csagola A, Lorincz M, Tombacz K, Spinu M, Tuboly T. Distribution and genetic diversity of porcine hokovirus in wild boars. Arch Virol. 2011;156:2233–9. http://dx.doi.org/10.1007/s00705-011-1125-6
5. Pan Y, Zeng Q, Zhu C, Hua X, Wang M, Pan K, et al. Frequency and characterization of porcine hokovirus (PHoV)

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in domestic pigs in eastern China. Arch Virol. 2012;157:1785–8.. http://dx.doi.org/10.1007/s00705-012-1350-7
6. Xiao CT, Gimenez-Lirola LG, Halbur PG, Opriessnig T. Increasing porcine PARV4 prevalence with pig age in the U.S. pig population. Vet Microbiol. 2012;160:290–6. http://dx.doi.org/10.1016/j.vetmic.2012.05.038
7. de Paula VS, Wiele M, Mbunkah AH, Daniel AM, Kingsley MT, Schmidt-Chanasit J. Hepatitis E virus genotype 3 strains in domestic pigs, Cameroon. Emerg Infect Dis. 2013;19:666–8.
8. Adlhoch C, Kaiser M, Loewa A, Ulrich M, Forbrig C, Adjogoua EV, et al. Diversity of parvovirus 4–like viruses in humans, chimpanzees, and monkeys in hunter–prey relationships. Emerg Infect Dis. 2012;18:859–62. http://dx.doi.org/10.3201/eid1805.111849
9. Tse H, Tsoi HW, Teng JL, Chen XC, Liu H, Zhou B, et al. Discovery and genomic characterization of a novel ovine partetravirus and a new genotype of bovine partetravirus. PLoS ONE. 2011;6:e25619. http://dx.doi.org/10.1371/journal.pone.0025619
10. Kamar N, Bendall R, Legrand-Abravanel F, Xia NS, Ijaz S, Izopet J, et al. Hepatitis E. Lancet. 2012;379:2477–88. http://dx.doi.org/10.1016/S0140-6736(11)61849-7

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Evaluation of 3 Electronic Methods Used to Detect Influenza Diagnoses during 2009 Pandemic

To the Editor: Conducting influenza surveillance in hospitals is imperative to detect outbreaks, inform infection control policy, and allocate resources (*1*). Hospital administrative data could be harnessed for this purpose (*2*,*3*) but are not currently used for infection surveillance because of data lag times. Influenza cases could be identified by using International Classification of Diseases, Tenth Revision, Clinical Modification (ICD-10-CM), codes within the discharge abstract, pharmacy, and microbiology laboratory information systems. Although these approaches are assumed to accurately identify influenza cases, this assumption has not been widely tested, especially during a pandemic. In this retrospective cohort study, we aimed to identify and evaluate 3 electronic methods of influenza case detection during 1 peak of influenza A(H1N1)pdm09.

With ethics board approval, we used the Ottawa Hospital Data Warehouse (OHDW) (Ottawa, ON, Canada) to identify 398 adult inpatients at the Ottawa Hospital during October–December 2009 who had cardiac, infectious, or respiratory disease diagnoses (ICD-10-CM codes: all J codes, A15–19, A37, A40, A41, A49, I26, I28, I50, I51.4, R57). OHDW is a relational database containing pharmacy, laboratory, and discharge diagnosis information for inpatients at Ottawa Hospital. We detected influenza in the following ways: influenza diagnosis in the discharge abstract database (DAD) (ICD-10-CM codes J09–J11); prescription for an antiviral drug (oseltamivir, zanamivir) in the pharmacy system; and a positive laboratory test during the hospital encounter (without specifying test type or specimen) in the laboratory system.

We assessed these case definitions against a criterion standard of influenza diagnosis on the hospital chart, determined by a physician reviewer blinded to the electronic values for the case definitions. We constructed 2 × 2 contingency tables for each classification method and calculated sensitivity, specificity, positive predictive value (PPV), and likelihood ratios using standard equations.

Influenza prevalence in this cohort was 13.6% (54/398) by our criterion standard. The proportion of male and female patients was equal, with a median age of 69 years (interquartile range 53–81 years). Median length of hospital stay was 6 days (interquartile range 1–12 days). A total of 77 (19.3%) patients were admitted to the intensive care unit, and 51 (12.8%) patients died in hospital. Two (0.5%) patients died with a primary diagnosis of influenza. The Table shows the performance characteristics of each influenza classification method against the criterion standard. The DAD-based influenza diagnosis algorithm was most accurate, with sensitivity of 90.7% (95% CI 79.7%–96.9%), specificity of 96.5% (95% CI 94%–98.2%), and PPV of 80.3%.

Our results demonstrate adequate correlation between ICD-10-CM coding for influenza in adults during a 3-month peak of the pandemic season within a single institution. Coding or interpretative errors were the probable cause of the 10% false-negative and 3% false-positive rates of ICD-10 coding for influenza on the DAD.

Classifying influenza by antiviral prescription was sensitive but less specific than clinical diagnosis. This finding could be explained by empiric antiviral prescriptions for